

Gout: Not Just for the Rich and Famous!

Everyman's Disease

Mary Kamienski

Gout is a painful form of arthritis that affects more than a million Americans annually. Using a case study, a discussion of the clinical onset, signs and symptoms, diagnosis, and treatment of acute and chronic gout is presented. The incidence and etiology of gout is presented, along with current diagnostic criteria and the most common differential diagnoses to consider. The current trends in diagnosing and treating the acute and chronic forms of gout are also addressed as well as some of the controversies and concerns about treatment options. The treatment plan and follow-up for the patient in the case presentation conclude the discussion.

History and Physical Examination

67-year-old man arrived in the urgent care center with a complaint of right foot pain that began approximately 6 days ago with no history of trauma. He reports the pain started at 3 out of 10 and is now 8 out of 10 on a pain scale of 0 to 10. He is unable to wear his work boots and has not been to work for 2 days. He states he normally sleeps from about 8 p.m. to 5 a.m. without awakening but has been awake every hour the last two nights because of the pain. He applied a heating pad to the

area, with minimal relief, and spent yesterday in a chair with his foot elevated. He admits he is more fatigued than usual but attributes it to a lack of sleep.

The patient is a Caucasian man with a history of hypertension. He has been taking clonidine 0.1 mg BID for more than 15 years and his blood pressure is usually 140/90. Two weeks before this visit, his primary care physician added hydrochlorothiazide 12.5 mg OD because his blood pressure was 148/96. Other significant medical history includes two episodes of renal calculus 10 years ago, successfully treated with lithotripsy and two episodes of diverticulitis more than 10 years ago, successfully treated with antibiotics. The patient denies any respiratory difficulties. He has never smoked. He also denies shortness of breath or dyspnea. Patient also denies any chest pain or extra heart beats.

He states his appetite has decreased in the past few days due to the discomfort in his foot and decreased activity. He has maintained his weight between 185 and 195 lbs for about 40 years. The patient drinks 2–3 vodka and tonic drinks every day for approximately 10 years and has not changed that pattern recently.

The patient believes he is in good health despite his chronic hypertension and this acute episode of foot pain. He maintains his weight, eats properly, and is physically active. He is still working full-time for the power and light company and has worked as a lineman for this company for 46 years. He has no plans to retire.

Physical examination reveals a 67vear-old white man sitting on the examination table. He is well developed, is normal weight, and appears younger than his stated age. His hygiene is good, and he is alert and cooperative. He was observed walking with difficulty into the examining room by the admitting nurse. The findings for the physical examination are listed in Table 1. In summary, the patient is a 67-year-old man in moderate distress related to the pain in his right foot. The physical examination is essentially normal, with the exception of the erythema, edema, and severe pain of the right great toe.

Primary Diagnosis

Gout presents with sudden intense joint pain that often starts in the early

Mary Kamienski, PhD, RN, APN, CEN, Assistant Professor, St. Peter's College, Nursing Department, Montville, NJ. sumption may contribute to gout, but they are not the main cause of this disorder. Gout is caused by an excess of uric acid in the body that may result from overproduction, underelimination, or increased intake of foods containing purines. These foods are metabolized to uric acid in the body. Certain red meats and organ meats, such as liver and kidneys, shellfish and anchovies, dried peas, and beans, are particularly high in purines. Uric acid crystals, which are related to sodium urate, can also form in the kidney, causing kidney stones (Goroll et al., 1995).

Primary gout is caused by either decreased excretion or increased production of uric acid. Hereditary underexcretion is the most common cause. Secondary gout is caused by acquired conditions, such as polycythemia vera, leukemia, and multiple myeloma, which cause overproduction of uric acid and chronic renal insufficiency; lead poisoning; diuretics; and low-dose salicylates, which are related to undersecretion of uric acid. When gout affects women, it is usually after menopause because estrogen promotes uric acid secretion. It is also strongly associated with hypertension, hyperlipidemia, and diabetes (Pittman & Bross, 1999).

Any sudden change in uric acid levels can trigger a gout attack. Factors that cause a rise in uric acid levels include the use of diuretics, alcohol, and low-dose aspirin. Lowering uric acid is attributed to sudden cessation of alcohol use, high-dose salicylates, or initiation of allopurinol or uricosuric drugs. Acute illness or surgery may alter uric acid levels and initiate an attack (Kidd & Robinson, 1999).

Physiologic and epidemiologic evidence link diuretic therapy with hyperuricemia. In a retrospective cohort study of 9249 individuals over the age of 65 years, the researchers concluded that use of thiazide diuretics in doses of 25 mg/day or higher is associated with a significantly increased risk for initiation of antigout therapy (Gurwitz et al., 1997).

Gout attacks can recur in the same joint. The initial attack may last several days to 2 weeks and may resolve without treatment. Some individuals may have a single attack, but 20% of patients will have at least a second attack, which may not occur for several years after the initial attack. In some



FIGURE 1. The joint involved in an attack of acute gout appears swollen and erythematous with periarticular involvement. The photo is from an article from the April 1, 1999, issue of *American Family Physician* titled Diagnosis and Management of Gout by Joel R. Pittman, Pharm D., and Michael H. Bross, M.D.

individuals, however, it may recur every few weeks. When gout attacks occur more often, they may involve more joints, last longer, and cause more severe symptoms. These repeated attacks could damage the affected joints (Pittman & Bross, 1999).

Polyarticular gout, with more than one joint involved, occurs in 70% of women, with hands being the most common site. Chronic gout may affect multiple joints and can be confused with rheumatoid arthritis or osteoarthritis (Kidd & Robinson, 1999).

Diagnosing Gout

Several other kinds of arthritis can mimic a gout attack (see Table 2 for a list of differential diagnoses). Because treatment is specific for gout, accurate diagnosis is essential. The most accurate diagnosis can be obtained by examining the joint fluid. Arthrocentesis of the affected joint produces fluid that is examined microscopically for sodium urate crystals that can be found in the joint fluid during an acute attack. Serum uric acid levels are often misleading and may be normal or even low. Additionally, uric acid levels can often be elevated in individuals without gout. Therefore, blood uric acid levels are a poor way to diagnose gout (Gurwitz et al., 1997). More commonly, the diagnosis of gout is based on patient history and examination of the affected joint.

Treatment

The literature is controversial, and there are several ongoing studies that indicate there is little reliable information based on randomized controlled trials on which to base treatment decisions in acute and chronic gout (Schlesinger, Baker, & Schumacher, Jr., 1999). Treatment options include nonsteroidal antiinflammatory drugs (NSAIDS), colchicine, and corticosteroids administered either intraarticularly, orally, or parenterally. Because of the common and unpleasant side effects from colchicine that include nausea, vomiting, and diarrhea, the drug may be discontinued before relief is achieved. Colchicine is contraindicated for use in patients with severe gastrointestinal (GI), renal, hepatic, or cardiac disorders. It should be used with caution in patients with impaired hepatic function or those who are elderly and debilitated. NSAIDs have therefore become the treatment of choice for most acute attacks of gout. (van Doornum & Ryan, 2000). Prompt intervention is usually more important than which option is used.

Chronic gouty arthritis is known as topachaceous gout and requires the

	TA	BLE	1		
Physi	cal	Exa	miı	nati	on

System	Findings
Vital signs	Blood pressure:142/92, right arm sitting; radial pulse is 78 with no irregularities noted, respirations normal depth and effort at a rate of 16. Temperature: 100.8°F tympanic.
Skin	Color is normal pink warm with multiple senile lentigines noted on the dorsum of both hands. Hair is brown with no thinning noted; mustache gra Patient is clean-shaven. Evidence of acne-related facial scarring.
Ear, nose, and throat	Head is normocephalic.
	Ears in normal position with no external tenderness. TM gray without injection. Rinne and Weber testing deferred.
	Nose: slightly bulbous without masses. Mucosa: pink with no discharge noted.
	Sinuses: (-) tenderness over frontal and maxillary sinuses.
	Throat: lips pink without lesions. Patient has upper and lower dentures. Buccal mucosa is pink. $(-)$ injection of posterior pharynx. Tonsils absent; uvula elevates in midline; gag reflex intact; tongue midline with no lesions.
	AP diameter 2:1; bilateral breath sounds clear with no adventitious sounds.
Chest	Breasts: mild gynecomastia; no masses or discharge
Heart	S _{1,} S(-) clicks or murmurs; PMI 5ICS
Vascular abdomen: bowel sounds normal in 4 quadrants; no guarding, rigidity, or tenderness; no visible pulsations	No edema of LE; LE warm to touch with all pulses (+); no point tenderness on palpation except in area of right great toe
Rectal	Deferred
Genitalia: uncircumcised male with normal genitalia	Uncircumcised male with normal genitalia
Lymphatic	(-) lymphadenopathy
Musculoskeletal	Right great toe is reddened, 2+ edema, skin intact with no drainage; full range of motion of all extremities
Neurologic	Patient is oriented to person, place, and time; cranial nerves 3, 4, 6-12 intact; gross sensory and motor strength intact; gait altered due to pain; DTRs (+)

TM, tympanic membrane; AP, anterior/posterior; LE, lower extremities; DTR, deep tendon reflexes.

morning hours. The affected joint is swollen and warm to touch and is red or purple. Gout is a common cause of acute monoarticular arthritis. Sodium urate crystals in the synovium incite a brisk inflammatory response. The condition is found most commonly among middle-aged and older men. Onset is rapid, peaking within 12 to 24 hours. The large toe is most often affected, but gout can also affect other joints in the leg (knee, ankle, and

foot) and, less often, joints in the arm (hand, wrist and elbow). The fingers are rarely involved, and the spine is almost never affected. Alcoholic binges or new use of thiazide diuretics may precipitate gouty attacks. A mild fever may even be present (Goroll, May, & Mulley, 1995).

This patient has a temperature of 100.8 °F. The location of the swelling at the metatarsophalangeal (MP) joint of the great toe, the sudden onset of

symptoms, and the recent addition of a thiazide diuretic to his medication regimen would support gout as the primary diagnosis for this patient.

What Is Gout?

Gout was once believed to be a disease of the rich and famous and has been known as the disease of kings caused by consuming rich food and fine wines. Diet and excessive alcohol con-

TABI	.E 2
Differential	Diagnoses

Diagnosis	Symptoms/Diagnosis/Treatment
Pseudogout	Pseudogout tends to occur in older patients with degenerative disease of the knees and is associated with hyperparathyroidism, hemochromatosis, and severe degenerative joint disease. It is mostly monoarticular but can be polyarticular. The finding of calcium pyrophosphate crystals in the synovial fluids is diagnostic. The site of the inflammation in this patient and the absence of other morbidity would tend to rule out this diagnosis. However, aspiration of synovial fluid and examination for calcium pyrophosphate would more definitively help to confirm or rule out this diagnosis.
Rheumatoid arthritis	Typically presents in subacute fashion with symmetric polyarthritis, although atypical forms include monoarticular and asymmetric disease. The most common sites are the wrists, proximal interphalangea (PIP) joints, and metacarpophalangeal (MCP) joints, but elbows, neck, hips, knees, ankles, and feet may also be involved. Extraarticular manifestation includes vasculitis, pulmonary nodules or interstitial fibrosis, mononeuritis multiplex, Sjögren's syndrome, and Felty's syndrome (splenomegaly, anemia, thrombocytopenia). Fatigue may dominate the early clinical presentation and precede onset of joint symptoms. Other systemic symptoms (fever, weight loss) are prominent in severe cases. Women are more often affected than men. Morning stiffness is almost universal. Rheumatoid factor (RF) is found in approximately 75% of cases. Diagnosis requires the presence of a constellation of findings. It is unlikely to be confused with monoarticular gout but may be confused with polyarticular (Tophaceous Gout) when tophi occur in a variety of sites and joints become deformed.
Septic arthritis	Occasionally results from direct extension from a site of trauma or from osteomyelitis. Eighty percent of the cases are monoarticular, with gram-positive organisms. <i>Enterobacteriaceae</i> can also cause septic arthritis, particularly in intravenous drug abusers, people who are immunocompromised, and people who are chronically ill. Fever, chills, and joint inflammation are usually prominent. A larger joint, such as the knee or hip, is most likely to be involved. Within 10 days of nongonococcal infection, radiographic evidence of cartilaginous and bony damage may appear. The location of the inflammation in this patient and the lack of compromising morbidity would rule out this diagnosis; however, X-ray of the joint would be necessary to definitively rule it out.
Ankylosing spondylitis	This disease begins insidiously and affects young men most severely, producing inflammation of spinal joints and connective tissue with subsequent calcification and ossification. Peripheral arthritis does occur, but more often in women. Large proximal joints, such as the hips or knees, are the predominant peripheral sites.
Psoriatic arthritis	This condition has both peripheral and axial forms. In the peripheral form, the distal interphalangeal (DIP) joints are most often affected and the nails are pitted. Psoriatic skin changes usually predate the onset of the arthritis but can be subtle. The spondylitic form of the disease may resemble ankylosing spondylitis, but the extent of spinal involvement is less.
Reiter's syndrome	This is primarily a disease of young men. Oligoarthritis, nongonococcal urethritis, and ocular inflammation are the defining features. Dermatologic features include shallow, painless ulcers of the glans penis and hyperkeratosis of the feet. Onset is sometimes associated with a recent dysentery or urethritis Joint involvement is asymmetric and of the lower extremities. Heel pain with plantar fasciitis and calcaneal periostitis is distinctive and mild spondylitis is common. Antinuclear antibody is negative.

use of antihyperuricemic agents to prevent progressive articular damage. Tophi often resolve after several weeks of therapy. Concurrent antiinflammatory therapy should be administered until visible urate deposits have resolved (Goroll et al., 1995).

Probenecid, sulfinpyrazone, and allopurinol can be used to prevent recurrent attacks (Pittman & Bross, 1999). In the presence of renal impairment, allopurinol, which decreases

the production of uric acid, is the treatment of choice for urate-lowering therapy, but doses should be adjusted for renal function. Urate-lowering therapy should only be used if recurrent episodes of gout occur despite aggressive attempts to reverse or control the underlying causes (van Doornum & Ryan, 2000).

Probenecid and sulfinpyrazone (uricosuric agents) work by blocking uric acid reabsorption in the proximal tubules of the kidney. They are fairly well tolerated but require ample fluid intake and multiple does per day, which may result in noncompliance with the therapy. These medications are contraindicated in clients with a history of renal lithiasis (Kidd & Robinson, 1999).

Uric acid levels should be monitored in patients with chronic gout before initiating long-term therapy based on the serious adverse reactions to xanthine oxidase inhibitors, which include rash, fever, hepatitis, eosinophilia, vasculitis, and renal insufficiency (Kidd & Robinson, 1999). Baseline blood urea nitrogen (BUN), serum lipid profile, and a complete blood count (CBC) with differential should be obtained before initiating antigout therapy.

Obesity, alcohol intake, and certain foods and medications are potential exacerbating factors and should be identified and modified (Pittman & Bross, 1999). Because only 10% of circulating purine is derived from dietary sources, restriction of dietary purine is not necessary. However, weight loss without fasting and reducing alcohol intake and binge drinking should be considered (Goroll et al., 1995.)

The treatment plan for this patient included rest and no heavy lifting or weight-bearing activity. He was advised to use no acetylsalicylic acid products and to increase his fluid intake to 64 oz (or eight 8-oz glasses) of water daily. The patient was advised to reduce his alcohol intake and to follow a low-fat diet. He was told not to work for 72 hours (Cash & Glass, 2000). Naproxen 750 mg by mouth followed by 250 mg every 8 hours was prescribed. The hydrochlorothiazide diuretic was discontinued, and the patient was instructed to continue to take Clonidine 1 mg as prescribed by his primary physician.

Based on the episodic nature of this visit and the patient's report that he has regular blood work ordered by his primary physician, no laboratory testing was done to confirm this diagnosis. The patient was advised to call his primary physician on Monday morning to discuss management of his hypertension without hydrocholorothiazide. He was advised to see his private physician or return to the center if any of the following signs and symptoms occurred:

- Fever >102.0°F;
- Rash;
- Swelling of extremities; and
- Vomiting/diarrhea (Cash & Glass, 2000)

Follow-Up

The patient was contacted the following day by telephone. He reported that the pain had decreased in severity to a 5/10 on a 0 to 10 scale. He was planning to call his private physician on Monday morning. A follow-up telephone call to the private physician confirmed that the patient had been seen and the treatment prescribed by the center was effective. Further followup determined that the patient had a recurrence approximately 2 months later. The patient accessed care immediately as previously instructed and this attack was effectively resolved within 48 hours with Naproxen. A follow-up visit was scheduled for 1 month to reevaluate the patient's status.

Acute attacks of gout require follow-up in 48 hours if the patient is not responding to treatment or symptoms have worsened. The patient should return in 4 to 8 weeks for further treatment or evaluation. Individuals with chronic gout should be seen annually for follow-up evaluation. Referral to an orthopaedic surgeon or rheumatologist should be made if the diagnosis is questionable, there is concern regarding a septic joint, or the patient is unresponsive to therapy (Kidd & Robinson, 1999).

Summary

The first descriptions of gout can be traced to the beginning of recorded medical history, yet controversies and unanswered questions remain regarding the diagnosis and treatment of this painful form of arthritis. The need for crystal identification in all cases is questionable, and even the methods of measuring uric acid levels is debatable. There is little reliable information based on randomized controlled trials to base treatment decisions in the acute and chronic form of gout (Schlesinger et al., 1999). However, more than 1 million Americans, primarily men, are affected by this painful and potentially disabling disease.

Although there is no cure for gout, some patients who have had multiple attacks of gout may need to take medications that lower uric acid blood levels. Proper education regarding the causes of the disease, the side effects of the medications, and the need for a quick diagnosis and prompt intervention should be emphasized.

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CE Test

Gout: Not Just for the Rich and Famous! Everyman's Disease



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- . Read the article on page 16.
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CE TEST QUESTIONS

GENERAL PURPOSE: To provide registered professional nurses with an overview of gout, including current treatment recommendations and preventative measures.

LEARNING OBJECTIVES: After reading this article and taking this test, you will be able to:

- 1. Discuss the risk factors for and manifestations of gout.
- 2. Outline treatment and education strategies for patients who have gout.
- 3. Describe factors for differentiating gout from related etiologies.

1. Which of the following manifestations are associated with gout?

- a. redness and swelling
- b. pallor and cool temperature
- c. gray color and clear drainage
- d. necrosis and loss of sensation

2. The inflammatory response in gout is a result of

- a. an acute infectious process.
- b. sodium urate crystals in the synovium.
- c. inflammation following acute trauma.
- d. an autoimmune process.

3. Which of these patients is at increased risk of developing gout?

- a. a 35-year-old female who has a family history of arthritis
- b. a 44-year-old obese female who smokes cigarettes
- c. a 55-year-old male who drinks alcohol
- d. a 77-year-old male who is bed bound

4. Initiation of which of these medications may precipitate a gout attack?

- a. clonidine (Catapres)
- b. nifedipine (Procardia)
- c. enalapril (Vasotec)
- d. hydrochlorthiazide (Esidrix)

5. Which of the following increases the risk for gout?

- a. multiple myeloma
- b. coronary artery disease
- c. asthma
- d. osteoarthritis

An over-the-counter drug that is associated with undersecretion of uric acid is

- a. tylenol.
- b. aspirin.
- c. benadryl.
- d. vitamin C.

7. According to Pittman & Bross (1999), which of the following is associated with gout?

- a. hyperlipedemia
- b. hypoglycemia
- c. chronic fatigue syndrome
- d. chronic renal failure

8. Recurrent gout attacks usually

- a. resolve without treatment.
- b. are of a shorter duration.
- c. may involve more joints.
- d. indicate treatment resistance.

9. Which of these sites is usually affected in women who have polyarticular gout?

- a. cervical spine
- b. hands
- c. shoulders
- d. knees

10. Which of these diagnostic tests is recommended to differentiate gout from other conditions?

- a. X-ray
- b. CT scan
- c. serum uric acid level
- d. arthrocentesis

Patients may discontinue prescribed colchicine before relief is achieved due to unpleasant side effects such as

- a. weight gain.
- b. headache.
- c. diarrhea.
- d. mood swings.

12. Colchicine therapy is contraindicated for patients who have

- a. diabetes mellitus.
- b. cirrhosis.
- c. osteoarthritis.
- d. epilepsy.

 13. The drug of choice for lowering urate levels in patients who have renal impairment is a. probenecid. b. allopurinol. c. sulfinpyrazone. d. primodone. 										a. finding of calcium pyrophosphate in the synovial fluids b. occurrence in men younger than 45 c. fever and weight loss d. involvement of the hip joint 17. Morning stiffness is characteristic of													
14. Which of these suggestions should you give to a patient who is taking probenecid for gout? a. "Drink 6 to 8 glasses of water daily." b. "Increase your intake of red meats while on this medication." c. "Perform weight-bearing exercises several times each day." d. "Avoid taking vitamin supplements for several weeks."											a. ankylosing spondylitis. b. Reiter's syndrome. c. septic arthritis. d. rheumatoid arthritis.												
15. Which of these laboratory tests should be obtained before initiating antigout therapy? a. blood urea nitrogen (BUN) b. serum albumin level c. serum calcium level d. prothrombin time									re														
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